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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/542,117	11/30/2005	Lily Yang	14507-50900	4006
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MORRIS MANNING MARTIN LLP 3343 PEACHTREE ROAD, NE 1600 ATLANTA FINANCIAL CENTER ATLANTA, GA 30326			BRISTOL, LYNN ANNE	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)
	10/542,117	YANG ET AL.
	Examiner Lynn Bristol	Art Unit 1643

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 07 November 2007.
- 2a) This action is FINAL. 2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 1-49 is/are pending in the application.
 - 4a) Of the above claim(s) 18-49 is/are withdrawn from consideration.
- 5) Claim(s) _____ is/are allowed.
- 6) Claim(s) 1-17 is/are rejected.
- 7) Claim(s) _____ is/are objected to.
- 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
 - a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)	4) <input type="checkbox"/> Interview Summary (PTO-413)
2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)	Paper No(s)/Mail Date: _____
3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)	5) <input type="checkbox"/> Notice of Informal Patent Application
Paper No(s)/Mail Date <u>7/19/06; 12/11/06</u> .	6) <input type="checkbox"/> Other: _____.

DETAILED ACTION

1. Claims 1-47 are all the pending claims for this application.

Election/Restrictions

2. Applicant's election without traverse of Group I (Claims 1-17) in the reply filed on 11/7/07 is acknowledged.
3. Claims 18-49 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected inventions, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 11/7/07.
4. A) Applicant's election without traverse of the species of tumor marker for survivin in the reply filed on 11/7/07 is acknowledged. Applicants comments and suggestion on p. 10 that the elected group is generic to all the species of Claim 2 (and all of the species should be examined) is not found persuasive.

Applicants have not set forth the rationale for examining any of the remaining species or any combination of the tumor marker species with the elected survivin tumor marker. Would one of skill in the art expect to find survivin mRNA in combination with the mRNA for the non-elected species of tumor marker all within in the same tumor sample? In other words, are all the species classified as being expressed in the same or different tumors (or are any overlapping in expression for certain tumors)? What is the relationship amongst the tumor markers?

Further, the examiner is not required to establish a search burden for an election of species requirement in a 371 application as in the present case. Nevertheless, the

search of all of the species of Claim 2 would be burdensome because Applicants have not established a correlation amongst any of the tumor markers or explained why or how the tumor markers are related, and searching all of the species is not co-extensive.

Upon further consideration, the Examiner agrees to the rejoinder of the tumor marker species for: cyclin D1, Her2/neu, a mutant K-ras, chymotrypsinogen, carcinoembryonic antigen and human chorionic gonadotropin.

The non-elected species of tumor marker in Claim 2 are withdrawn from examination.

B) Applicants' election of the oligonucleotide of SEQ ID NO:2 (for survivin) without traverse is acknowledged. Because of the rejoinder of tumor marker species for cyclin D1, Her2/neu, and a mutant K-ras, the oligonucleotide sequences of SEQ ID NOS: 1 and 9 (survivin); SEQ ID NOS: 3 and 4 (cyclin D1); SEQ ID NOS: 5 and 6 (Her2/neu); SEQ ID NOS: 7, 8, 11, 12 and 13 (K-ras mutant gene) have also been rejoined for examination. The oligonucleotide of SEQ ID NO:10 corresponds to GAPDH, which as addressed below, is not a tumor marker. Nevertheless the sequence is joined for examination.

5. Claims 1-17 are all the pending claims under examination for this application.

Information Disclosure Statement

6. The IDS' from 7/19/06 and 12/11/06 have been considered and entered. The examiner's initialed 1449 forms are attached.

Claim Objections

7. Claims 2, 3 and 16 are objected to because of the following informalities:

- a) Claim 2 recites numerous typographical errors in the spelling of the tumor marker species. For example, "basic fiborblast growth factor" should recite "fibroblast"; "prostate, specific antigen" should recite "prostate specific antigen"; "alpha-fetalprotein" should recite "alpha-fetal protein".
- b) Claim 3 recites "the sample taken from" which appears to be a typographical error. Do Applicants mean "the sample is taken from..."? Appropriate correction is required.
- c) Claim 16 recites "targets-the tumor-marker a K-ras mutant gene" which appears to be a typographical error. Do Applicants mean "targets the tumor marker for mutant K-ras"? Claim 2 defines the tumor marker as "a mutant K-ras."

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

8. Claims 1-17 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

- a) Claims 1-17 recite the limitation "the target sequence" in elements iii) and iv) of Claim 1. There is insufficient antecedent basis for this limitation in the claim. Is the target sequence different from the tumor marker mRNA?

b) Claims 1-17 are indefinite for the limitation "the hybridization of the target sequence under suitable hybridization conditions" in Claim 1 because the phrase "suitable hybridization conditions" is not defined in the specification or the claims. The phrase does not describe any particular set of conditions and one of skill in the art would not be able to determine what conditions, and thus, what molecules Applicant intended the claim to encompass. Since the specification does not provide an unambiguous definition for the term, the metes and bounds of the claimed invention cannot be determined.

c) Claims 1-17 are indefinite for the recitation "a probing nucleobase sequence" in Claim 1 because it is not clear what is meant by the term "probing." The term implies an action or activity that must be performed by or with the sequence. Do Applicants mean "a nucleobase sequence probe"? Otherwise, a step in the method process appears to be missing.

d) Claim 9 is indefinite for the recitation "the oligonucleotide is SEQ ID NO:10" because the oligonucleotide is designed for targeting GAPDH (see Table 2 in the specification) which is not a tumor marker as required by the method claims. GAPDH is a housekeeping gene and the mRNA is generally used as a positive control for mRNA and to access the integrity of mRNA for a sample in an mRNA assay (see for example Abstract of Wen et al. Cancer Gene Ther. 7(11):1469-1480 (2000)). Have Applicants identified a new role for GAPDH as a tumor marker?

e) Claim 13 is indefinite for reciting improper Markush group language. See MPEP 803.02.

f) Claim 16 is indefinite for the recitation "the oligonucleotide targets the tumor marker a K-ras mutant gene" because the method of claim 1 is drawn to detecting tumor marker mRNA with the oligonucleotide, whereas Claim 16 is drawn to targeting the gene for a mutant K-ras. Is a different method step required in Claim 16 that requires gene targeting over targeting mRNA?

Priority

9. The priority claim to U.S. Provisional Application No. 60/439,771 (filed 1/13/2003; "771") for the method of using molecular beacon probes for detecting survivin mRNA (SEQ ID NOS: 1, 2 and 9), cyclin D1 mRNA (SEQ ID NOS: 3 and 4), Her/neu mRNA (SEQ ID NOS: 5 and 6), K-ras mutant mRNA (SEQ ID NOS: 7, 8, 11, 12 and 13) and chymotrypsinogen is acknowledged. The U.S. Provisional Application '771 does not teach or suggest a molecular beacon for carcinoembryonic antigen or human chorionic gonadotropin but the international application, PCT/US04/00755 (filed 1/13/2004) does support the tumor marker for the method assay.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

- (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
- (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

human chorionic gonadotropin

10. Claims 1, 2, 3, 4 and 5 are rejected under 35 U.S.C. 102(a) as being anticipated by Span et al. (Clin. Chem. 49(7); 1074-1080 (July 2003)).

Claims 1, 2, 3, 4 and 5 are drawn to a method for detecting tumor marker mRNA in a sample of cells by treating the sample with an oligonucleotide comprising at least one linked energy donor moiety and at least one linked energy acceptor moiety where the oligonucleotide forms a stem-loop hairpin and the donor and acceptor moieties are separated by a portion of the nucleobase sequence and where the oligonucleotide targets the tumor mRNA, and a target sequence is detected, identified or quantitated by hybridization with the oligonucleotide wherein a change in signal from the donor or acceptor moiety is correlated with the target sequence (Claim 1), the tumor marker is human chorionic gonadotropin (Claim 2), the sample is a tissue (Claim 3), the tissue is a biopsy from breast (Claim 4) or the tissue is a frozen section (Claim 5).

Span discloses a method for detecting human chorionic gonadotrophin- β -3, 5 and 8 mRNAs in human breast tissues using molecular beacon probes, for example, the design of which is described on p. 1075, Col. 2, ¶4: "A probe (5'-FAM-cgcttccagtccaagcg-TAMRA-3', where FAM is 6-carboxyfluorescein and TAMRA is 6-carboxytetramethylrhodamine) that specifically annealed with transcripts from the hCG- β -3, 5 and 8 genes was designed essentially according to guidelines for Molecular

Beacons described by Tyagi and Kramer (ref. 13) "... "However to obtain a smaller loop sequence, only one arm, complementary to five nucleotides at the 5' end of the hCG- β sequence was attached 3'." Biological samples were fresh frozen tissue from a tumor bank obtained from surgically resected breast tumors (p. 1075, Col. 1, ¶3) and a section would have been used on which to perform the assay (p. 1075, Col. 2, ¶2). The assay relies on a closed tube format using real-time fluorescence reverse transcription PCR where the data are quantitative and linear over a wide range of template concentrations (p. 1079, Col. 2, ¶2). Concentration or amount of the hCG- β mRNAs in the tissues was determined following PCR amplification with the probe and found to have independent prognostic value for relapse free survival in sporadic breast cancer patients (Figure 1). Thus Span reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for human chorionic gonadotropin mRNA in breast tumor tissue sample.

Her2/neu

11. Claims 1, 2, 3 and 14 are rejected under 35 U.S.C. 102(a) as being anticipated by Vijayanathan et al. (Antisense & Nuc. Acid drug Devel. 12:225-233 August 2002)).

The interpretation of Claims 1-3 is discussed supra. Claim 2 is drawn to the tumor marker for Her2/neu, Claim 3 is drawn to the sample being a tissue and Claim 14 is drawn to the oligonucleotide targeting the tumor marker Her2/neu.

Vijayanathan discloses using two molecular beacons (MB) to identify Her2/neu expression where Her2/neu overexpression is considered to be a negative prognostic

marker for some tumors (p.226, Col. 1, ¶2), The first MB is composed of a 6-nt complementary flanking region at both ends and a 15-nt loop region comprising an antisense sequence capable of hybridizing to the AUG translational start site of the Her2/neu 2 mRNA. The second MB probe was a 32-mer composed of a 7-nt complementary flanking sequence at both ends and an 18-nt loop region. The donor and acceptor were fluorescein and 4-([4'-(dimethylamino) phenyl]azo)benzoic acid, respectively. Vijayanathan teaches that the method is qualitative because the MB can "readily distinguish targets differing by a single nucleotide" and quantitative because the MB can detect nanomolar concentrations of target substrate and further because the assay can be performed within a cell in real-time setup (p. 231, Col. 1, ¶1). Thus Vijayanathan reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for Her2/neu mRNA in cell samples from tumors.

K-ras mutant

12. Claims 1, 2, 3, 4 and 16 are rejected under 35 U.S.C. 102(a) as being anticipated by Singer et al. (Am. J. Pathol. 160(4):1223-1228 (April 2002)) as evidenced by Vogelstein et al. (PNAS 96:9236-9241 (1999)).

The interpretation of Claims 1-4 are discussed *supra*. Claim 2 is drawn to the method where the tumor marker is mutant K-ras, Claim 3 is further drawn to the sample from blood or urine, and Claim 16 is interpreted as being drawn to the oligonucleotide targeting the K-ras mutant gene (as discussed under 112, 2nd *supra*). The breadth of

generic claim 1 encompasses any tumor marker mRNA from any biological sample from any tumor, thus Singer anticipates the claims.

Singer teaches examining K-ras mutations at codon 12 and 13 using digital PCR and molecular beacons (MB) (p. 1225, Col. 2, ¶3) in paraffin-embedded tissue samples of ovarian serous tumors. Singer incorporates by reference the methodology of Vogelstein for digital PCR using MB for detecting K-ras mutants in colorectal cancer cell lines. Vogelstein teaches the nucleotide sequence for MB red and MB green for K-ras mutant at p. 9237, Col. 1, ¶2. Vogelstein teaches that the method is useful for detecting small numbers of mutant containing cancer cells in for example blood and urine (p. 9236, Col. 1, ¶1). Singer discloses detecting and identifying the K-ras mutants, and by incorporation to Vogelstein, quantitating tumor gene expression levels. Thus Singer as evidenced by Vogelstein reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for K-ras mutant gene in ovarian tumor tissue sample, blood or urine.

Survivin and GAPDH

13. Claims 1, 2, 3, and 10 are rejected under 35 U.S.C. 102(b) as being anticipated by Wen et al. (Cancer Gene Ther. 7(11):1469-1480 (2000)).

The interpretation of the claims 1-3 is discussed supra. Claim 2 is drawn to the tumor marker for survivin and Claim 10 is drawn to the oligonucleotide targeting survivin.

Wen discloses methods for measuring and quantitating survivin mRNA with molecular beacon probes labeled with a reporter fluorescent dye [FAM (6-carboxy-fluorescein)] at the 5'- end and a quencher fluorescent dye TAMRA (6-carboxy-tetramethyl-rhodamine) at the 3'- end when added to a real-time PCR reaction. Wen teaches that when the probe is intact, the fluorescence emission of the reporter is quenched due to the physical proximity of the reporter and quencher fluorescent dyes (p. 1471, Col. 2, ¶1). Wen teaches detecting survivin and GAPDH mRNA from biopsy samples which are obtained from xenografted human breast cancer cell lines grown in mice *in vivo* as established tumors (p. 1447, Col. 2, ¶2). Thus Wen reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for survivin (and house keeping gene for GAPDH) in human breast tumor tissue sample.

K-ras mutant, survivin and chymotrypsinogen

14. Claims 1, 2, 3, 4, 7, 8, 9, 10, 16 and 17 are rejected under 35 U.S.C. 102(e) as being anticipated by Bao et al. (US 20060127940; published 6/15/06; filed 6/25/02).

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 102(e) might be overcome either by a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not the invention "by another," or by an appropriate showing under 37 CFR 1.131.

The interpretation of Claims 1-4 and 16 is discussed supra. Claim 2 is drawn to the tumor marker for chymotrypsinogen, Claim 4 is drawn to the tissue from the pancreas, Claim 7 is drawn to the sample from pancreatic juice, and Claim 8 is drawn to quantification of the tumor marker by FACS-scan analysis. Claims 9 and 17 are drawn to the oligonucleotide of SEQ ID NO: 7, 8, 11 and 12.

Bao discloses methods for detection of nucleic acids comprising K-ras and survivin (and p53, p16, DPC4, or BRCA2) as indicating the presence of a genetically associated disease, such as certain cancers [0046] using hairpin stem-loop molecular beacon (MB) probes that hybridize on the subject nucleic acid, e.g. mRNA, to generate an observable interaction for the identification and quantification of tissue and cell-specific gene expression levels (Abtrsact). Bao discloses where for the MB the donor moiety can be for example a 6-Fam fluorophore or the acceptor moieties can be Cy-3, ROX or Texas Red [0041-0042]. See the attached sequence search alignment for SEQ ID NOS: 7, 8, 11 and 12 against the K-ras mutant oligo MB of Bao. Example 3, Bao discloses the method where oligo probes for K-ras and survivin are both mixed into a hybridization reaction to detect mRNA in samples such as in blood, pancreatic juice and pancreatic tissue samples [0186]. Bao discloses measuring K-ras, survivin and chymotrypsinogen in pancreatic cancer cells [0198]. Bao discloses using a FACS Vantage SE cell sorter (Becton-Dickinson) to sort out the cancer cells in the mixture in suspension because of its very high detection sensitivity, capability of 5 color analysis and sorting, wide flexibility of excitation wavelengths, and cross beam laser compensation for separation of overlapping excitation [0198-0199]. Thus Bao reads on

the method steps of detecting, identifying and quantitating the presence of the tumor marker for survivin, chymotrypsinogen and K-ras mutant in blood, pancreatic tissues and pancreatic juices where quantification is accomplished by FACS.

cyclin D1, Her2/neu and carcinoembryonic antigen

15. Claims 1, 2, 3, 4, 6, 12, and 14 are rejected under 35 U.S.C. 102(e) as being anticipated by Harbeck et al. (US 20060084056; published 4/20/2006; priority to 2/13/2002).

The interpretation of Claims 1–4 is discussed supra. Claim 2 is drawn to the tumor marker for cyclin D1, Her2/neu and carcinoembryonic antigen, Claim 3 is drawn to the sample from breast lavage, aspiration or needle biopsy, Claim 12 is drawn to the oligo targeting cyclin D1 and Claim 14 is drawn to the oligo targeting Her2/neu.

Harbeck teaches measuring mRNA levels for breast tumor markers for urokinase-type plasminogen activator (uPA) and plasminogen activator inhibitor-1 (PAI-1) in combination with cyclin D1 and carcinoembryonic antigen to facilitate the classification of subjects into high and low risk subjects [0067; 0195] using biological samples like any body fluid of the subject including blood, serum, plasma, milk, urine, saliva, pleural effusions, synovial fluid, spinal fluid, tissue infiltrations and tumor infiltrates, cells, tissues and tissue extracts [0021; 0196] and core needle biopsy and body fluid aspiration from breast, where reagents for binding a nucleic acid (e.g., a mRNA, a spliced mRNA, a cDNA, or the like) include complementary nucleic acids, for example, molecular beacon probes [0207]. Harbeck discloses a comparison of the

prognostic value using the above markers to known Her2/neu tumor marker in scoring breast cancer [0243]. Thus Harbeck reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for cyclin D1, carcinoembryonic antigen and Her2/neu for breast cancer.

K-ras mutant, survivin and chymotrypsinogen

16. Claims 1, 2, 3, 4, 7, 8, 9, 10, 16 and 17 are rejected under 35 U.S.C. 102(e) as being anticipated by Bao et al. (USPN 7297494; published 11/20/07; priority to 6/25/2001 for K-ras mutant molecular beacon)

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 102(e) might be overcome either by a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not the invention "by another," or by an appropriate showing under 37 CFR 1.131.

The interpretation of Claims 1, 2, 3, 4, 7, 8, 9, 10, 16 and 17 is discussed supra.

Bao discloses methods for detection of nucleic acids comprising K-ras and survivin (and p53, p16, DPC4, or BRCA2) as indicating the presence of a genetically associated disease, such as certain cancers [0046] using hairpin stem-loop molecular beacon (MB) probes that hybridize on the subject nucleic acid, e.g. mRNA, to generate an observable interaction for the identification and quantification of tissue and cell-specific gene expression levels (Abtrsact). Bao discloses where for the MB the donor

moiety can be for example a 6-Fam fluorophore or the acceptor moieties can be Cy-3, ROX or Texas Red [0041-0042]. See the attached sequence search alignment for SEQ ID NOS: 7, 8, 11 and 12 against the K-ras mutant oligo MB of Bao. In Example 3, Bao discloses the method where oligo probes for K-ras and survivin are both mixed into a hybridization reaction to detect mRNA in samples such as in blood, pancreatic juice and pancreatic tissue samples [0186]. Bao discloses measuring K-ras, survivin and chymotrypsinogen in pancreatic cancer cells [0198]. Bao discloses using a FACS Vantage SE cell sorter (Becton-Dickinson) to sort out the cancer cells in the mixture in suspension because of its very high detection sensitivity, capability of 5 color analysis and sorting, wide flexibility of excitation wavelengths, and cross beam laser compensation for separation of overlapping excitation [0198-0199]. Thus Bao reads on the method steps of detecting, identifying and quantitating the presence of the tumor marker for survivin, chymotrypsinogen and K-ras mutant in blood, pancreatic tissues and pancreatic juices where quantification is accomplished by FACS.

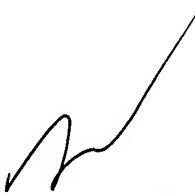
Conclusion

17. No claims are allowed.
18. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Lynn Bristol whose telephone number is 571-272-6883.
The examiner can normally be reached on 8:00-4:00, Monday through Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on 571-272-0832. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

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